THE USE OF OXYGEN IN COMATOSE STATES*

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THE essential factors related to the maintenance of normal tissue oxygen tension and to the adequate removal of carbon dioxide have been evaluated from measurements obtained on arterial blood and expired air. The adequate removal of carbon dioxide constitutes an important aspect of the respiratory gas exchange. The arterial oxygen saturation may be maintained near normal by the administration of oxygen, yet the carbon dioxide removal may be hampered by inadequate ventilation or interference with the transfer of gases in the lungs. The two main factors to be considered in oxygen therapy are: 1) an inspired oxygen partial pressure (pO_2) of sufficient magnitude to saturate the arterial blood (normal 96 to 98 per cent[1]) and 2) an adequate ventilation providing sufficiently uniform alveolar aeration to wash out the carbon dioxide. This latter is determined from measurement of total ventilation in liters per minute, the partial pressure of carbon dioxide in the arterial blood (pCO_2) and arterial blood pH. If the arterial blood hemoglobin is from 96 to 98 per cent saturated and the carbon dioxide partial pressure and pH are within the normal range, the respiratory gas exchange is adequate.

The direct determination of the tension of oxygen (pO_2) and carbon dioxide (pCO_2) in arterial blood[2] has been most helpful in studying the transport of oxygen to the blood. The difference between the mean oxygen partial pressure of inspired air (150 mm.Hg average at sea level) and that of the alveolus is referred to as the *aeration gradient*, and the difference between mean oxygen partial pressure of the alveolus and that of the arterial blood is designated as the *transfer gradient.*[3] The term *transfer gradient* has been used because this value deals with the movement of oxygen from the gaseous state in the alveolus either to combination with hemoglobin or to solution in the blood plasma.

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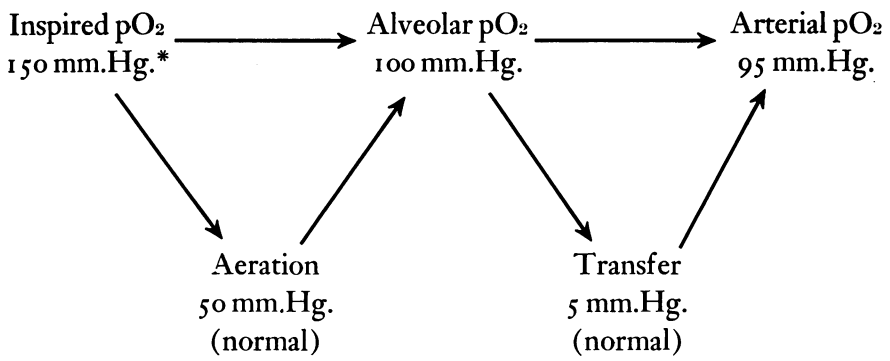
OXYGEN TRANSPORT IN THE LUNGS



Elevation of the transfer gradient designates changes in the alveolar-arterial oxygen gradient from all causes, including the true diffusion difficulty with an increased resistance of the pulmonary membrane.

Factors interfering with the oxygen respiratory gas exchange are primarily those related to mixing, dilution, diffusion and distribution. Decreased ventilation (reduction in vital capacity, maximal breathing capacity or both), increased residual air (pulmonary emphysema with the mixing and dilution problem), impaired diffusion due to increased resistance in the pulmonary membrane (as seen in acute pulmonary edema) and distribution (unequal alveolar aeration and perfusion) are the factors most often responsible for lowering the arterial oxygen tension and saturation.

Increased residual air, decreased ventilation, retained secretions and bronchospasm increase the aeration gradient and lower the alveolar pO_2 and arterial oxygen saturation. Hyperventilation will lower the aeration gradient and raise the alveolar pO_2. Any factor increasing the resistance to the movement of oxygen across the pulmonary membrane (as pulmonary edema) lowers the arterial pO_2 and increases the transfer gradient. Gross venous admixture such as occurs in congenital cardiac anomalies and arteriovenous fistulae produces very high transfer gradients (often over 50 mm.Hg) and arterial blood unsaturation even when the subject breathes 100 per cent oxygen. Unequal alveolar aeration and perfusion (the distribution factor) are a common cause of elevation of the transfer gradient. Fibrosis produces various degrees of

* Ambient air breathing at sea level, barometric pressure 760 mm.Hg.

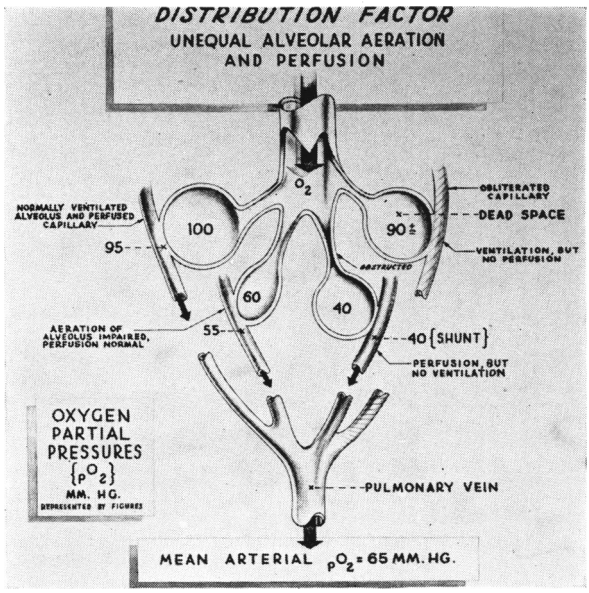
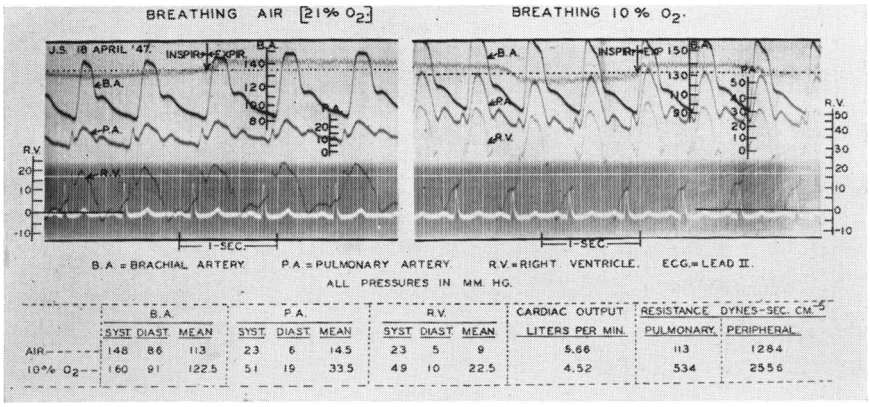


Fig. 1—A schematic diagram indicating the disturbed relationship between alveolar aeration and perfusion, as produced by pulmonary fibrosis, emphysema, retained secretions, bronchospasm, consolidation, pulmonary atelectasis or depression of respiration. Intermittent positive pressure breathing increases the alveolar pO_2 in those alveoli which have impaired aeration but are still perfused with blood. Intermittent positive pressure breathing has no effect on the oxygen transfer in alveoli which are perfused but nonaerated (unless some alveoli are opened up with the increased inspiratory pressure) or alveoli which are ventilated but not perfused. Elevating the inspired oxygen tension (pO_2) corrects for the unequal alveolar aeration, increases the arterial pO_2 to the normal range and compensates for small shunts due to perfusion with no alveolar aeration. In large shunts, however, such as those in the pulmonary circulation, even 100 per cent oxygen does not completely saturate the arterial blood.

impairment of the air circulation (loss of elasticity in the lung and narrowing of the bronchiolar lumina) so that although perfusion may be present, ventilation is inadequate or absent (Fig. 1). Alveoli which are perfused but nonaerated present circulatory pathways which act as small shunts and lower the arterial oxygen saturation (Fig. 1). The magnitude of such shunts can be calculated.[4,5] The blood flow to the alveolar capillaries may be obliterated to a greater or lesser extent, resulting in ventilation without perfusion, and the alveoli so involved are functional dead space. Hyperventilation can improve the situation only in alveoli poorly ventilated but still perfused with blood. The distribution factor is a common involvement in chronic pulmonary conditions with fibrosis, but this factor may occur with various degrees of obstruction to alveolar aeration as in atelectasis, consolidation of the lung, re-



	B.A.			P.A.			R.V.			CARDIAC OUTPUT	RESISTANCE DYNES-SEC. CM.$^{-5}$	
	SYST	DIAST	MEAN	SYST	DIAST	MEAN	SYST	DIAST	MEAN	LITERS PER MIN.	PULMONARY	PERIPHERAL
AIR	148	86	113	23	6	14.5	23	5	9	5.66	113	1284
10% O_2	160	91	122.5	51	19	33.5	49	10	22.5	4.52	534	2556

Fig. 2—Photograph of pressure tracings showing the effect of low oxygen (10 per cent) breathing upon pulmonary artery blood pressure. Tracings of pressure in the pulmonary artery and in the right ventricle were taken through a double lumen catheter and recorded by a Hamilton manometer.[7] The maximal increase in pressure in the pulmonary artery was noted within five minutes after the low oxygen breathing was started. Some patients were observed for as long as twenty minutes on 10 per cent oxygen; the pulmonary artery pressure remained at the constant high level, but decreased to normal within five minutes after the low oxygen was removed.

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tained secretions or fluid and bronchospasm.

Acute hypoxia of short duration has been found to elevate pulmonary artery pressure in animals[6] and in man[7] (Fig. 2). Pulmonary vascular resistance was increased by acute hypoxia, but cardiac output in man as determined by the direct Fick method, was not increased.[7] The comatose patient should be promptly treated with oxygen to maintain a normal arterial oxygen saturation and thus prevent the increase in pulmonary vascular resistance which accompanies acute hypoxia and cyanosis. Cyanosis is a poor guide to the actual degree of hypoxia present. The recently perfected quantitative oximeter[8] provides a method for determining the arterial oxygen saturation which is more rapid and probably as accurate as the difficult and laborious Van Slyke method.[9]

Increasing the inspired partial pressure of oxygen (pO_2) may be adequate to produce a normal arterial oxygen saturation if there are no large areas of shunting such as occur in pneumonia with consolidation and in congenital defects. Maintenance of a normal arterial oxygen saturation by increasing the inspired pO_2 does not assure adequate removal of the carbon dioxide, because the minute volume ventilation

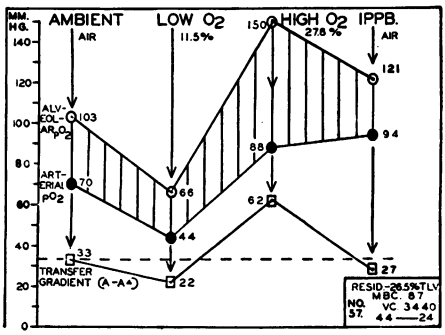

Fig. 3

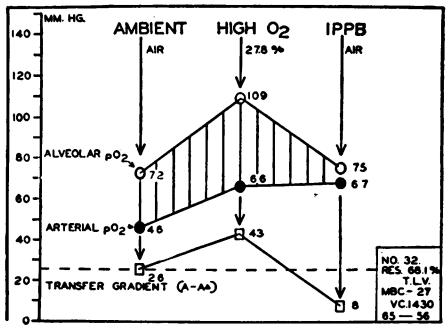

Fig. 4

Fig. 3—Diagram showing changes in alveolar and arterial pO_2 with low and high oxygen levels and intermittent positive pressure breathing in a patient with fibrosis but no pulmonary emphysema. The transfer gradient (the difference between alveolar and arterial mean pO_2) was greater than 30 mm.Hg. on ambient breathing. Note that the arterial pO_2 was increased with intermittent positive pressure breathing more than with high oxygen, although the alveolar pO_2 and transfer gradient were lower. Intermittent positive pressure breathing produces a more uniform aeration of those alveoli poorly ventilated by ambient breathing but still perfused with blood, and thus increases the arterial pO_2 more in proportion than the elevated alveolar pO_2 level of ambient breathing on high oxygen. Alveolar pO_2 calculated by the indirect method.[34]

Fig. 4—Diagram of changes produced in alveolar and arterial pO_2 with high oxygen breathing and intermittent positive pressure breathing in a patient with a far advanced degree of pulmonary emphysema. The transfer gradient (26 mm.Hg. on ambient breathing) was increased on high oxygen breathing and decreased to a normal level with intermittent positive pressure breathing, although the alveolar pO_2 was only 75 mm.Hg. with intermittent positive pressure breathing as compared to 109 mm.Hg. with high oxygen breathing, and the arterial pO_2 was not increased with the latter. Intermittent positive pressure breathing by increasing the aeration in the poorly ventilated alveoli on ambient breathing corrected the unequal alveolar aeration almost completely in this patient, and as there was very little perfusion difficulty, the transfer gradient assumed almost normal value.

may be grossly inadequate and many areas of the lungs poorly areated (as in atelectasis, pulmonary fibrosis, bronchospasm and pulmonary edema). That the use of intermittent positive pressure breathing provides a more uniform aeration of those alveoli which are poorly aerated but still perfused with blood is shown by the increase in the arterial oxygen saturation and decreased transfer gradient (Figs. 3 and 4).[10] The arterial pO_2 and oxygen saturation were increased more with intermittent positive pressure breathing on compressed air than with high oxygen (27.8 per cent) during ambient breathing (Figs. 3 and 4).[11]

Adequate removal of carbon dioxide depends on the minute ventilation and the uniformity of the alveolar aeration and perfusion. If the respiration is depressed, the carbon dioxide increases and the pH of the

TABLE I—OXYGEN BREATHING IN PULMONARY EMPHYSEMA (AMBIENT) ON A DEMAND SYSTEM

Case	*Residual Air Percent of Total Lung Volume*	*Arterial Blood*			
		On Air		*On 99.7% O_2 (10 min.)*	
		pCO_2**	pH	pCO_2**	pH
D.M.*	54.5	66	7.42	80	7.32
J.S.*	56.5	70	7.36	89	7.30
W.B.	61.5	47	7.46	64	7.39
L.M.	64.6	56	7.41	76	7.38

* Cardiac disease present.
** mm.Hg.

TABLE II—OXYGEN BREATHING IN OPERATIVE PROCEDURES

Thoracoplasty	*Arterial Blood*		
	*pO_2***	*pCO_2***	*pH*
Ambient Breathing, Air	98	37	7.48
During Anesthesia, controlled breathing—IPPB* type—with curare.	549+	69	7.32
Ambient Breathing, one hour postoperative—oxygen by B.L.B. mask.	520+	56	7.34

* Positive pressure only during inspiration-expiration at atmospheric pressure.
** mm.Hg.

arterial blood decreases (respiratory acidosis). The long-continued use of high oxygen levels, as 100 per cent oxygen in controlled anesthesia or in pulmonary emphysema (Tables I and II), lowers the arterial blood pH by elevating the arterial pCO_2 either through a decrease in ventilation or unequal alveolar aeration. Hydrogen ion concentrations as low as 6.90 have been observed in arterial blood during controlled anesthesia on patients receiving 100 per cent oxygen for three hours or more.[12] The arterial blood pH decreases gradually, and the longer the exposure on 100 per cent oxygen with the controlled anesthesia, the lower the pH. In patients with hypoxia as a result of inadequate ventilation the pCO_2 is elevated and the arterial blood pH decreased.

Patients needing artificial respiration or supplement to ambient breathing have an elevated arterial blood pCO_2, so that the further use of carbon dioxide in oxygen (as 5 or 7 per cent) is not physiologic.[13] The use of 5 per cent carbon dioxide only increases the acidosis of the apneic subject in need of artificial respiration. Studies on arterial blood before treatment have revealed decreased oxygen tension, arterial oxygen unsaturation, increased carbon dioxide content, increased pCO_2 and decreased arterial blood pH.[13] It is a popular misconception, largely due to the influence of Henderson,[14] that carbon dioxide mixtures are of value in the treatment of respiratory depression of asphyxia. Mixtures of carbon dioxide as high as 20 per cent have been advocated for use in artificial respiration on the assumption that a respiratory center too depressed to respond to 5 per cent carbon dioxide would respond to a higher concentration.[14] In the asphyxiated patient the blood carbon dioxide level is above normal so that the carbon dioxide no longer acts as a respiratory stimulant but in fact may be a depressant. In dogs subjected experimentally to severe, acute anoxia, 5 per cent carbon dioxide produced respiratory depression, and 15 per cent carbon dioxide produced even more profound respiratory and circulatory depression when administered in the same stage of hypoxia.[15]

Present concepts indicate that there is no rational basis for the use of carbon dioxide in asphyxia and that such a procedure is not only contraindicated but dangerous.[13, 15-19] Recently an experimental study was reported[18, 20] indicating that even in carbon monoxide poisoning, the addition of CO_2 to oxygen does not appear advantageous, oxygen alone being preferable. The Council on Physical Medicine and Rehabilitation of the American Medical Association and the American National Red Cross recommend that oxygen alone be used in resuscitators or inhalators instead of mixtures of oxygen and carbon dioxide.[21] It is of interest that in Fulton's recent (1949) "Textbook of Physiology"[22] the use of carbon dioxide gas mixtures in resuscitation is still advocated, although the present evidence reveals little rationale for the addition of carbon dioxide to gas breathing mixtures for use by patients with depressed breathing and hypoxia. Patients with chronic pulmonary disease and an advanced degree of impairment of pulmonary function with hypoxia may occasionally become delirious or unconscious when subjected to high concentrations of oxygen, a phenomenon attributable to the presence of a high pCO_2 with a respiration that is

being primarily maintained reflexively from the carotid sinus by the hypoxia stimulation; when the stimulation is suddenly removed by the high concentration of inspired oxygen the reflex control of respiration is decreased. The respiratory center has apparently become tolerant to carbon dioxide and thus the increase in arterial pCO_2 brought about by the decrease in ventilation while breathing the high oxygen mixture has no further stimulating effect. The decrease in arterial blood pH and the increase in the arterial pCO_2 appear primarily responsible for producing the delirious or unconscious state. I observed a patient at Bellevue Hospital with bullous emphysema and marked cyanosis who became unconscious within ten minutes after being placed on 100 per cent oxygen; when the patient was returned to air breathing with the appearance of cyanosis, consciousness returned. In such a patient, acclimated to chronic hypoxia of long duration, the concentration of oxygen breathed should be increased gradually.[23, 24] However, when a patient with far advanced bullous emphysema becomes acclimated to a high concentration of oxygen, discontinuance of the oxygen is extremely difficult.

Helium reduces the resistance of breathing air in and out of the lungs, and may relieve subjective dyspnea. The minimal helium-oxygen mixtures should be 70 per cent helium and 30 per cent oxygen, for if a 80 per cent helium and 20 per cent oxygen mixture is used, arterial blood measurements have revealed that in some cases the oxygen saturation* decreases (Fig. 5). The breathing may be easier with helium, but the oxygen saturation of the arterial blood may not be higher. The oxygen percentage in commercially supplied helium-oxygen tanks varies considerably; one tank of a supposedly 20 per cent oxygen and 80 per cent helium mixture delivered to our laboratory actually contained only 18.68 per cent oxygen, a lowering of sufficient magnitude to be significant to the patient with far advanced emphysema or depressed respiration. By reason of its great diffusibility, helium is a difficult gas to control, and gas mixtures containing helium are maintained at fixed concentrations only by the utmost care. Helium is also an inert foreign gas and the use of a 20 per cent oxygen and 80 per cent helium mixture after 10 minutes may lower the arterial oxygen saturation in emphysematous patients with adequate external minute ventilation. The use of from 30 to 40 per cent oxygen in the helium-oxygen gas mixture

* Determined from O_2 content and capacity on Van Slyke apparatus.[9]

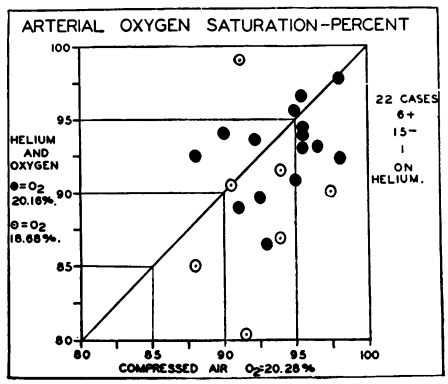

Fig. 5—Comparison of the resting arterial oxygen saturation while breathing compressed air with that obtained while breathing a helium-oxygen mixture through a demand regulator. These data were obtained on 22 cases of emphysema and fibrosis (most of the patients had anthracosilicosis). Note that in 10 cases the arterial oxygen saturation was slightly higher during the breathing of compressed air than during the breathing of the helium and oxygen mixture, although the oxygen percentage of both gas mixtures was essentially the same. When the arterial oxygen saturations of the 7 patients breathing the helium-oxygen mixture containing 18.68 per cent oxygen (this tank was supposed to contain 20 per cent oxygen and 80 per cent helium) were compared to those in the patients who breathed compressed air, there was a decrease in five cases, an increase in one case, and no change in one case. If a helium-oxygen gas breathing mixture is used, the oxygen should be increased to a minimum of 30 per cent.

appears to be the preferable procedure when helium is indicated to reduce breathing resistance in the dyspneic patient.

The question of irritation from the prolonged use of high oxygen concentrations may be a factor for consideration in the comatose patient. Observations made with the pneumatic balance respirator during long use of oxygen did not reveal any demonstrable irritating effect on the lung, as far as could be determined.[25] In one instance a postmortem examination of the lungs revealed no irritation of the alveoli or bronchi after three days of continuous intermittent positive pressure breathing through a tracheal catheter of 100 per cent oxygen by an apneic patient suffering from cerebral concussion following a blow on the head with a baseball bat. The greatest danger from the prolonged use of high oxygen concentrations in ambient breathing appears to arise from the decreased ventilation and reduction in uniformity of alveolar aeration, a condition which, in the presence of retained secretions, tends to promote the development of atelectasis in the more dependent portions of the lungs. The addition of a small amount of an inert gas (helium or nitrogen) is effective prophylaxis against the development of atelectasis,

which probably is the most serious complication of extended ambient breathing of 100 per cent of oxygen at atmospheric pressure. Substernal distress has been reported [26] in normal subjects breathing high oxygen concentrations for twenty-four hours. Breathing dry oxygen during a simulated ascent to high altitude in a low pressure chamber may irritate the throat, producing coughing and mild substernal discomfort somewhat similar to chokes (a manifestation of aeroembolism). Since the inspired pO_2 is elevated to a very high level with 60 per cent oxygen (Barometric pressure −47 mm.Hg. × 60 per cent = effective inspired pO_2), it is unnecessary to use 100 per cent oxygen in ambient breathing in comatose patients to saturate the arterial blood. The use of from 60 to 70 per cent oxygen mixture will protect against atelectasis and yet provide a high enough inspiratory pO_2 for maintenance of good arterial oxygen saturation in most cases, but such a mixture does not necessarily provide adequate ventilation.

Common methods of increasing oxygen tension and saturation in the arterial blood include the use of: the oxygen tent, the nasal catheter, the continuous flow oxygen system with a mask (BLB and OEM) and the demand oxygen system (Mine Safety Appliance and Scott Aviation Corporation). The demand oxygen system (unless provided with a diluter arrangement on the regulator) provides essentially the same oxygen concentration to the mask as in the supply tank of compressed gas; hence the demand system should not be used continuously on comatose patients by ambient breathing over prolonged periods of time unless the tank mixture has an inert gas dilutant such as nitrogen or helium. The Drinker type respirator can be used to give artificial respiration with oxygen therapy provided by a catheter, mask or demand system. The prolonged use of the Drinker type respirator predisposes to the formation of atelectatic areas in the lungs and loss of muscle tone, often complicated by the development of bronchopneumonia and pulmonary edema. Bennett[27] has designed a cycling valve to supplement the Drinker respirator with intermittent positive pressure breathing, a procedure that has decreased complications in bulbar poliomyelitis patients and has saved lives.

Intermittent positive pressure breathing (IPPB) consists of an active inflation of the lungs during inspiration under positive pressure from a cycling valve with a maximal peak pressure at the mouth adjustable from 0 to 30 centimeters of water. After the valve cycles, expiration

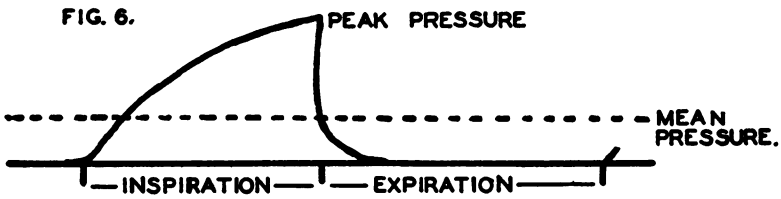

Fig. 6—Mask pressure curve illustrating the time-pressure relationship previously described and discussed as type III.[28] The type III mask pressure curve is asymmetrical, with a pressure gradually increasing during inspiration and rapidly dropping to atmospheric level early in expiration. Expiratory time equals or exceeds inspiratory time, and the inspiratory mean mask pressure is high as compared to the expiratory mean mask pressure, which is very low (most of the mean mask pressure for the entire respiratory cycle being exerted during the inspiratory phase). This type of mask pressure curve (type III) provides a peak pressure which is effective in overcoming breathing resistance and maintains adequate ventilation with a minimal mean mask pressure without decreasing cardiac output in man.[13, 28]

occurs as a passive deflation produced primarily by the elasticity of the lungs and the wall of the chest. The cycling of the respirator valve follows the patient's pattern of breathing.

Factors to be considered in using IPPB are related to the shape and duration of the mask pressure applied at the mouth; namely, the time relationship of inspiration and expiration, the cycling rate and the magnitude of pressures applied (maximal, minimal and mean). The most desirable type of pressure to apply to the mask or through an intratracheal tube is a mask pressure curve that is asymmetrical, gradually increasing during inspiration and rapidly dropping to atmospheric level early in expiration. Expiratory time is equal to or greater than inspiratory time, and most of the mean mask pressure is exerted during the inspiratory period (Fig. 6), previously described as type III mask pressure curve.[28] This type of intermittent positive pressure breathing provides a high peak mask pressure effective in overcoming breathing resistance, yet the mean mask pressure for the entire respiratory cycle is low. No deleterious effects on the pulmonary circulation (Fig. 7), either on blood pressure or cardiac output,[28] were noted when the type III IPPB was used. IPPB has been found useful in the treatment of acute pulmonary edema,[29] in the maintenance of adequate ventilation in depressed respiratory states,[13] in giving breathing exercise with more uniform distribution of simultaneously administered aerosols,[30] in promoting bronchial drainage,[31] and in providing more uniform alveolar

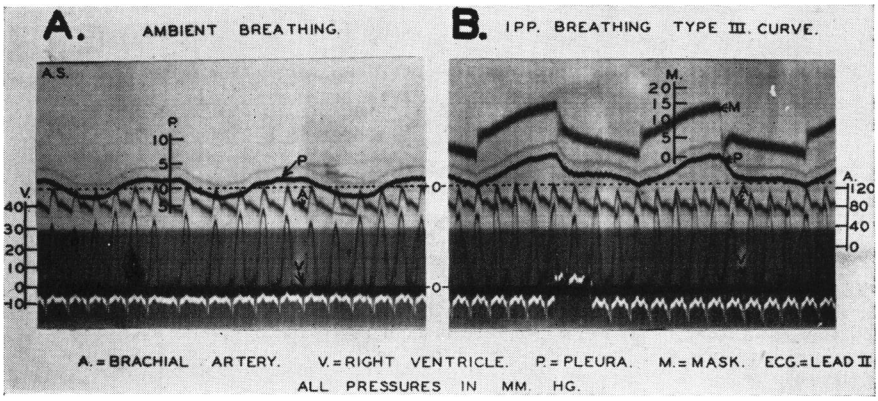

Fig. 7—A photograph of pressure tracings during ambient and respirator breathing, using type III mask pressure curve on an essentially normal subject. (A) Pressure tracing during ambient breathing. From above downward: pleural, brachial artery and right ventricle. (B) Pressure tracing while breathing with the respirator. From above downward: mask, pleural, brachial artery and right ventricle.

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aeration,[3] with protection against the development of atelectasis.[27] No apparent advantage has been found from the use of negative pressure in part of the respiratory cycle,[13] and the potential danger of negative pressure in patients with threatened pulmonary edema would seem to make IPPB the method of choice.

The use of intermittent positive pressure breathing for prolonged periods does not produce distention of the lung or increase the amount of residual air even in patients with a far advanced degree of emphysema.[32] The instantaneous flow rate of the respiratory appliance may be too low to meet the needs of the acute asthmatic and cardiac patient (Fig. 8).[29]

The Pneophore* is an automatic, pressure-sensitive, cycling device for giving IPPB for therapeutic use. This apparatus provides adequate minute ventilation volumes even in apneic subjects, and may be combined with a nebulizer for the simultaneous administration of an aerosol during the IPPB.[30] The nebulizer attachment on the Pneophore makes a valuable emergency apparatus for giving adrenergic drugs with oxygen in depressed respiratory states because the intake of the drug from the large pulmonary absorptive area is rapid. Since the in-

* Manufactured by Mine Safety Appliance Co., Pittsburgh, Penna.

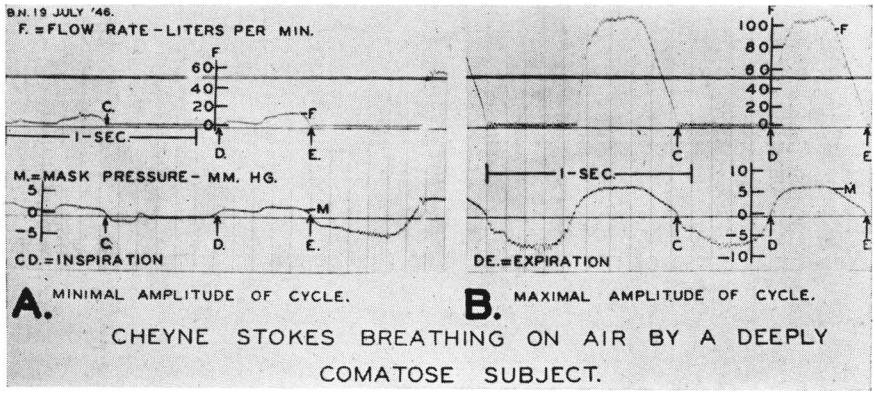

Fig. 8—A photograph of a tracing of instantaneous flow rates and mask pressure curves on a patient with Cheyne-Stokes breathing. (A) During period of minimal breathing, the maximal instantaneous flow rate was approximately 10 liters per minute. (B) During period of maximal breathing, the instantaneous flow rate exceeded 100 liters per minute. The flow capacity of respirator apparatus must be greater than the maximal instantaneous flow rate of the patient at all times in order to prevent the development of a greater negative pressure during inspiration with subjective dyspnea in the conscious subject.

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stantaneous flow rate is approximately 50 to 60 liters per minute, this apparatus should not be used on acute decompensated cardiacs or asthmatics. The Demand Pneophore has a demand regulator to provide additional flow capacity in case the instantaneous flow rate of the "Burns" type cycling valve[33] used on the standard Pneophore is inadequate. The Demand Pneophore is suitable for use on all types of patients. The Bennett type cycling valve* based on flow characteristics rather than pressure[25] is very satisfactory for administering IPPB, especially for the combined use with the nebulizer in giving aerosols. This apparatus provides high instantaneous flow, has an atmospheric mean expiratory pressure at the mouth and is easy to clean and maintain because there is no rebreathing through the cycling valve. The cycling valve follows the patient's pattern of breathing and the instantaneous flow rate is high (well over 100 liters per minute). Operation of this unit is economical, as oxygen may be used to enrich the compressed air as indicated. This apparatus is useful in treatment for all types of depressed breathing, including postoperative anesthetic cases, because it simultaneously increases ventilation and inspired oxygen, blows off car-

* Manufactured by J. J. Monaghan Company, Inc., Denver, Colorado.

bon dioxide and protects against atelectasis.

Electrocardiographic studies made after prolonged periods of IPPB with 100 per cent oxygen and potent bronchodilator drugs (Vaponefrin, Isuprel) revealed no significant changes. However, in several instances when compressed air was used instead of 100 per cent oxygen, depression of the RS-T segments and inversion of the T waves were noted with the drugs mentioned.[31] The electrocardiogram was not altered by the use of neosynephrine in the nebulizer with the IPPB.

Conclusions

1. The two essential factors for consideration in the maintenance of normal respiratory gas exchange in comatose subjects are: (1) an inspired pO_2 of sufficient magnitude to saturate the arterial blood and (2) adequate ventilation to wash out the carbon dioxide effectively, thus preventing the development of respiratory acidosis. Administration of high concentrations of oxygen controls the first factor in most cases, but on ambient breathing alone the second factor is often inadequately handled.

2. Unequal alveolar aeration and perfusion (the distribution factor) produced by fibrosis, bronchospasm, atelectasis, consolidation, retained secretions or depressed respiration or any combination of these, usually constitute the major impairment present in the comatose patient in need of oxygen therapy.

3. Acute hypoxia increases pulmonary vascular resistance and increases the load on the right side of the heart.

4. Carbon dioxide should not be used with the oxygen in respirators, resuscitators or insufflators for treating asphyxia because the use of 5 per cent carbon dioxide only increases the acidosis in apneic subjects in need of artificial respiration.

5. The preferred minimal oxygen-helium mixture is 30 per cent oxygen and 70 per cent helium.

6. The development of atelectasis appears to be the greatest danger from the prolonged use of high oxygen concentrations during ambient breathing, but repeated intervals of intermittent positive pressure breathing (IPPB) appear to offer effective prophylaxis.

7. For prolonged use by ambient breathing in comatose patients, 60 to 70 per cent oxygen mixed with an inert gas protects against atelectasis and in most cases saturates the arterial blood adequately, however,

this mixture does not necessarily provide adequate ventilation. A lowering of the arterial blood pH below 7.35 reveals the presence of severe uncompensated respiratory acidosis due to an inadequate minute ventilation volume.

8. IPPB provides more uniform aeration of alveoli perfused with blood, but previously poorly aerated, increases minute ventilation volume with maintenance of adequate ventilation during respiratory depression, lowers arterial pCO_2, protects against atelectasis, promotes bronchial drainage and provides a useful method in the treatment of acute pulmonary edema.

9. IPPB can be administered without alterations of cardiac hemodynamics (lowering blood pressure, or cardiac output). It provides adequate ventilation in the comatose patient so that respiratory acidosis is readily controlled. Several types of respirators for producing IPPB are described. The instantaneous flow rate of these devices should be at least 100 liters per minute.

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